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AUTHOR Grantham, Madeline Kay
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ABSTRACT

This paper explores the historical origins of attention deficit disorder/attention deficit hyperactivity disorder (ADD/ADHD) as a neurological disorder, current neurological and genetic research concerning the etiology of ADD/ADHD, and implications for diagnosis and treatment. First, ADD/ADHD is defined and then the origins of ADD/ADHD as a neurological disorder are traced in a review of the periods of 1800 to 1950, 1950 to 1970, 1970 to 1990, and 1990 to 1999. The review then goes on to consider implications of neuro-imaging technology, a look at frontal lobe and parietal lobe involvement, and implications of the neuro-transmitter dysfunction theory. Examination of ADD/ADHD and genetics reviews family studies, twin studies as a basis for ADHD genetic etiology, and adoption studies. Discussion of other genetic discoveries notes new research concerning the thyroid hormone receptor and a dopamine transporter gene. The final section considers assessment and treatment of ADD/ADHD including current assessment procedures, traditional treatment, and other treatment approaches. One of the five conclusions is that neurological studies involving brain scans indicate brain peculiarities in approximately 70 percent of the children diagnosed with ADD/ADHD. (Contains 39 references.) (DB)

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Running Head: ADHD: NEUROLOGY AND GENETICS

Etiology of Attention Disorders: A Neurological/Genetic Perspective

Madeline A. Grantham

Mississippi State University

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Abstract

This paper explored the historical origins of ADD/ADHD as a neurological disorder, current neurological and genetic research concerning the etiology of ADD/ADHD, implications for diagnosis and treatment, and conclusions relevant to the study of the etiology and treatment of ADD/ADHD. Although environment must always be considered a factor, results of research indicated that genetic and neurological studies might lead to different methods for the identification and treatment ADD/ADHD (Aman, et.al. , 1998, Barkley, 1998; Greenhill, 1998; Gibbs , 1998; Gross, 1997; Ingersoll & Goldstein, 1993; Rowe, et.al., 1998; Wender, 1987; Zametkin et. al, 1990).

There has been much controversy over the frequent diagnosis of ADD/ADHD in children and the etiology of this disorder. ADD/ADHD has been described as a major public health problem (Greenhill, 1998). A nationwide estimate indicated that ADD/ADHD prevalence exists among 2%-9% of the school age population (about 2 million children). Furthermore, 30%-50% of mental health referrals of children are for ADD/ADHD (Barkley, 1998; Gross, 1997). Diagnosis of this disorder is based on the characteristics outlined in the DSM IV (Diagnostic Statistical Manual --Fourth Edition). A list of these may be found in the appendix. For the purpose of this literature review, ADD/ADHD has been defined as a disorder which is neurobiologically based and consists of the 3 inappropriate observable behaviors of inattention, impulsivity and/or hyperactivity; a disorder that affects learning as well as behavior (Barkley, 1995). This paper explored the history of a neurological etiology of ADD/ADHD, current research in these areas, and implications for the future diagnosis , treatment, and study of the etiology and treatment of ADD/ADHD.

Origins of ADD/ADHD as a Neurological Disorder

1800-1950

ADD/ADHD, as a neurological disorder, has an extensive historical perspective. "Fidgety Phil" was a character in stories written in the 1800's by a German family doctor. Most authors credit the beginnings of a behavioral diagnosis with the work of Still and Meyer in 1902, who explored brain trauma and attempted to tie it to unruly behaviors (Barkley, 1981). Behavior problems were relegated to a secondary position by teachers until an encephalitis epidemic in 1924 in England when scientists tried to establish a relationship between the disease and the resulting hyperactive behavior of children with the disease (Barkley, 1981).

Links were also established between hyperactive behaviors and head wound injuries in adults and animals. During the 1930's experiments on monkeys demonstrated that damage to or deficiencies in the frontal lobes preceded overactive and/or uninhibited behavior (Robertson, 1994). In 1937, Bradley, while working with encephalitis patients, detected an unusual response to Benzedrine. While using amphetamine to improve blood pressure and relieve headaches, he discovered that the behaviors of difficult children improved while on the medication. In the United States, this resulted in attempts to find a link between problems with children at school and brain dysfunction. There was a dramatic increase in studies on the use of amphetamines with children exhibiting hyperactive and impulsive behaviors. (Schachar, 1986). In the 1940's, Strauss, in cooperation with Lehtinen and Werner, coined the label "Minimal Brain Damage". This term was used to link brain damage at birth with inattentive, hyperactive and impulsive behavior (Small, 1982).

1950-1970

During this time period, the new terminology used to describe children with hyperactivity behavior problems were the phrases hyperkinetic syndrome and the hyperactive child syndrome (Barkley, 1998). The demonstrated effectiveness of some drugs upon undesirable behaviors prompted the treatment of these syndromes towards the medical field (Ross & Ross, 1976). In 1962, the characteristics of hyperactivity, learning deficits in the presence of normal intelligence, impulsivity, distractibility, short attention span, and frequent abnormal EEG's were included among the characteristics of children diagnosed with brain dysfunction (Gross , 1977).

1970-1990

During the years 1971-1980, the term hyperactivity was emphasized. Some researchers felt that technology avenues, such as brain scanning, did not definitively back up a link between

brain damage and unruly behaviors (Small, 1982). The emphasis on brain damage shifted to an emphasis on brain dysfunction as more interest was placed on inattention and impulsivity. The medical-disease model emphasized medication for use as a form of social control. It was theorized that the growth of pharmaceutical companies, scientific explorations on medical treatment for behavior problems and government control of drug use were the major factors that contributed to the emphasis on brain dysfunction and medication treatments. (Conrad , 1976) Treatment of hyperactive behavior became the exclusive realm of the doctor and reinforced the medical-disease status of hyperactivity.

In 1980, the DSM-III(Diagnostic Statistical Manual – third edition) renamed Hyperkinetic Disorder to Attention Deficit Disorder, with and without hyperactivity (Jordan, 1992).Wender (1987) described ADD as an inborn temperamental difference in a child. Wender noted that treatment affects the severity of the problem, but cannot cause the problem . Weiss (1984) was one of the few dissenters who argued for equal consideration of biological, psychological and social factors.

1990-1999

Current researchers utilize technological tools to attempt to demonstrate that ADD/ADHD is neurologically based and is genetically and biologically transmitted (Barkley, 1998; Gross, 1997; Reader, Harris, Schuerholtz, & Denckla, 1994). The interpretations of these technological studies vary.

Russell Barkley , who emphasizes a medical-disease model, has dominated the research of the 90's into the etiology of ADHD/ADD. Barkley describes ADD/ADHD as a developmental failure in the "brain circuitry that underlies inhibition and self control . . . which impairs other important brain functions crucial for maintaining attention, including immediate

rewards for later, greater gain (Barkley, 1998; Aman, Roberts & Pennington, 1998; Greenhill, 1998; Gross, 1997) . In 1990, PET (positron emission topography) brain imaging studies were performed on children and adults diagnosed with ADHD. The studies indicated that 3 areas of the left brain show patterns that demonstrate the failure of the brain to use sugar (brain glucose) fast enough to maintain normal thought patterns and muscle responses, triggering a pattern described as an ADHD behavior pattern (Zametkin, Nordahl, Gross, King, Semple, Rumsey, Hamburger, Cohen, 1990). Even though the DSM-IV, in 1994, indicated that no definitive laboratory tests have been established for diagnosing ADD/ADHD, Russell Barkley continues to emphasize genetics, brain injury and/or abnormal brain development as possible etiologies for this syndrome. He emphasizes self-control and interest span rather than attention span and has presented the new label of *Developmental Disorder of Self Control* (Barkley, 1995). This scientific, neurological history provides a basis for the current usage of scientific technology to explore a neurological etiology of ADHD.

Implications of the Neuro-Imaging Technology

Neuro-imaging is an important technological tool used for studying brain functioning. Researchers have used this technology to explore which regions of the brain might malfunction in patients with ADD/ADHD. Imaging studies have suggested that the prefrontal cortex, part of the cerebellum and at least two clusters of the basal ganglia (nerve cells deep in the brain) are involved in the production of ADD/ADHD symptoms (Barkley,Grodzinsky, & DuPaul , 1992 Castellanos, et. al).

A recent study utilized Magnetic Resonance Imaging (MRI) scans on 576 ADHD diagnosed boys, ages 5 to 18. Results showed that the right prefrontal cortex and the 2 basal ganglia known as the caudate nucleus and the globus pallidus, were significantly smaller in

children diagnosed with ADHD in comparison with normal children in the control group. The researchers also noted that the vermis region of the cerebellum was smaller in ADHD diagnosed children (Castellanos , 1997). This is significant because these are the areas thought to be involved with regulating attention. It is thought that the right prefrontal cortex edits behavior, resists distractions and provides an awareness of self and time. The caudate nucleus and globus pallidus switch off automatic response to allow time for careful deliberation and coordination of neurological input among the various regions of the cortex (Barkley, 1998). The study described the prefrontal cortex as the "steering wheel" and the caudate and globus as the accelerator and brakes. The braking or inhibitory function is likely what is impaired, creating the ADD/ADHD symptoms. (Castellanos,1997).

Another study noted that functional resonance imaging provides an extremely effective method for mapping brain function and structure. (Zametkin , et. al. , 1998). Six completed structural imaging studies utilizing Photo Emission Topography (PET), reported differences between the ADHD brain and the normal control brain. A decrease was noted in the brain metabolism of the basal ganglia and equal symmetry. The corpus calosum seemed to be involved. However, the authors avoided drawing strong conclusions and suggested that future research should investigate the degree of involvement.

These studies indicated a possible linkage between ADHD symptoms and the structure of the brain. It was suggested that the degree of involvement and the portions of the brain involved could possibly be discerned through replication studies (Zametkin, et. al. , 1998).

A Look at the Frontal Lobe and Parietal Lobe Involvement

These parts of the brain influence the functions of learning and memory, wakefulness and attentiveness, hunger and thirst, impulsivity, explosiveness and rage, stress, sexuality and negative social behaviors such as belligerence, uncooperativeness and anger (Aman, et. al, 1998;

Barkley, 1998). Recent research indicated the involvement of the frontal lobes and right parietal lobe as a possible influence on ADD/ADHD symptoms. The researchers also indicated that it is equally important to determine why there are decrements in performance in the task domains and how these are related to brain functioning, rather than trying to explain the underlying deficit in ADD/ADHD in terms of the frontal lobe theory or parietal lobe theory (Aman, et. al, 1998; Barkley, et. al., 1992). Identification of the portions of the brain involved in this disorder could lead to treatment of these areas through medication and other creative procedures which would eventually be developed through research.

Implications of the Neuro-transmitter Dysfunction Theory

Gross (1997), an internist and endocrinologist who shifted his clinical practice exclusively to the diagnosis and treatment of ADD/ADHD, viewed this disorder as a neurological problem. He defined the disorder as "a complex of clinical dysfunctions, which at the neuroscience level, are associated with reduced effectiveness of neurotransmitter hormone action and/or synaptic abnormality in various centers of the brain (1997)." Gross proposed that the presence of ADD/ADHD symptoms, as presented in the DSM-IV, are not the only problems associated with ADD/ADHD. He has attempted to link neurotransmitter and synaptic abnormalities in the brain to academic underachievement, sleep problems, irritability, negative feelings and attitudes and dyslexia problems.

The medical practice data presented indicated that the ADD/ADHD individual, driven by an ADD brain chemical imbalance, does not understand the reasons for his/her need for adventurous or driven behavior and is surprised when disaster strikes. (Gross, 1997). Dr. Gross supported his theory by noting the effectiveness of medication on ADD/ADHD neurological dysfunctions and indicating that medications have measurable actions on the neurotransmitter

synaptic mechanism. The neurotransmitter/synaptic on-off communication process takes place between a large number of brain nerve cells over short, measurable periods of time. An interruption or problem with neurotransmitter/synaptic activity could create problems with an individual's thoughts, feelings and body movements that are the expression of this activity. Gross (1997) feels that the limbic system or primitive part of the brain located near the center of the brain is responsible for the ADD./ADHD disorders. Medication treatment is implicated if this is the problem area.

Researchers investigating ADD/ADHD are diligently searching for a possible neurological etiology. Brain imaging studies are now able to show the structure of the brain in very fine grained detail, and this new functional MRI procedure also carries less risks than the PET (Barkley, 1998). This new technology could help confirm or disprove these theories.

ADD/ADHD and Genetics

Another area of study in the search for the etiology of ADD/ADHD is the area of behavioral genetics. Genetic discoveries and theories, which relate to this disorder, will be presented in this section.

Behavior Genetics: Nature vs Nurture

Researchers concerned with the etiology of ADD/ADHD must consider the issue of genetics vs environment. The research methodologies used to support a genetic etiology over environment consist primarily of family studies, twin studies and adoption studies.

Family Studies

Most behavior geneticists believe that there is no single gene for intelligence, personality, behavior or even height, but rather such characteristics are polygenetic, that is influenced by multiple genes. Behavioral geneticists emphasize that while both genes and environment are

important and unique to the individual, shared environment is not important (Eyesenck, 1990). Although many studies indicated a high prevalence of ADD/ADHD among males, female members also have an increased risk of the disorder indicating a possible genetic tie rather than a gender basis for diagnosis. (Zametkin, et. al. 1998). Faulty genetics as an etiology of ADHD could possibly be verified through the study of relatives of children identified as ADD/ADHD. Siblings of children with ADHD are between 5 and 7 times more likely to develop the syndrome than children from unaffected families. Children of a parent with ADD/ADHD have up to a 50% chance of experiencing the same problem (Biederman, 1998; Barkley, 1998). In one study, 58% of 84 children who met criteria for ADHD , were also the children of adults with this disorder. These results indicated support for the validity of possible strong familial etiological risk factors for children of adults with ADHD (Biederman,1998). Other studies have indicated that relatives of ADD children are at greater risk for the disorder than the relatives of control children (Cantwell, 1992; Quinn, 1997).

Twin Studies as a Basis for ADHD Genetic Etiology

Studies of identical and fraternal twins have possible significant implications for genetics as a possible etiology of ADD/ADHD. One study found a significantly higher incidence of ADD in identical twins over fraternal twins, suggesting a genetic predisposition to ADD (Quinn, 1997). Another study used a model-fitting strategy to estimate genetic and environmental contributions to the core behavioral dimensions associated with ADHD. The study involved 576 sets of twin boys ages 11 and 12. Data was collected through teacher rating scales and maternal interviews, using the DSM-III criteria for ADHD. Data was obtained for 194 monozygotic (identical) twins and 94 pairs of dizygotic (fraternal twins). The study concluded that genetic factors are etiologically significant in the expression dimensions of ADHD such as inattention,

impulsivity and hyperactivity. Bivariate analyses indicated a significant correlation between the 2 dimensions that was genetically mediated (Sherman, Iacono, McGue, 1997).

It has been estimated that 40% of ADHD kids have a parent who has the trait and 35% have a sibling with the problem. When the sibling is an identical twin, the chances rise to between 80-92 percent (Barkley, 1998; Wallis, 1994). A large twin study was conducted at the University of Oslo in conjunction with the University of Southampton in England. This study involved 526 identical twins who inherit the same exact genes and 389 fraternal twins who were no more alike genetically than other siblings. The study concluded that ADHD has a heritability approaching 80%, meaning that up to 80% of the differences in attention, hyperactivity and impassivity between people with ADHD and those without the disorder, can be explained by genetic factors (Gjone and Skukndt, 1997).

The newest twin study performed at the University of New South Wales, Australia, analyzed 1,983 families with twins and other children aged four to twelve years old. When one twin had ADHD, there was a 91% chance for the identical twin to have it. Growing up in the same environment had only a 13% heritability effect on ADHD, and ADHD was the highest disorder of heritability over other types of familial behavior disorders. The researchers concluded that ADHD is extreme behavior that will vary genetically throughout the entire population (Levy, Hay, McStephen, Wood, Waldman, 1997).

Adoption Studies

Wender (1997) stated that "adoption constitutes a scientifically well designed, but unintended social experiment by which one can determine whether an individual adoptee's psychopathology is correlated with nature or nurture." One study found that a history of childhood hyperactivity was 12 times more common in the first-degree and second-degree

relatives of the adopted and biological ADHD children evaluated. It was also discovered that 33% of the biological parents of hyperactive children received a diagnosis of alcoholism, sociopathy or hysteria versus 5% among the adoptive parents (Cantwell, 1975). In this study, biological/genetic behavioral traits were strongest in the biological relationships over the adoptive relationships, indicating a possible genetic tie to behavioral disorders.

Other Genetic Discoveries

Thyroid Hormone Receptor

It has been discovered that some people have a different receptor for the thyroid hormone. One study showed that 70 to 80 percent of people with this rare difference exhibit ADD/ADHD symptomology. (Zametkin, et. al, 1998). Another study showed that 70% of the children and 50% of adults with generalized resistance to thyroid hormone, also met the criteria for ADHD (Quinn, 1997). Weiss (1993) indicated that there was a connection between a unique receptor for the thyroid hormone and ADD/ADHD characteristics. Future research needs to investigate this different receptor and/or a resistance to thyroid hormone as an indicator of a possible genetic influence on the etiology of ADD/ADHD.

Dopamine Transporter Gene

More recently, the tracer fluoro-dopa was used to determine if there were differences in dopaminergic uptake in the brains of adults and adolescents with ADHD. Differences were noted when compared to normal brains. ADHD patients have responded positively to medications which inhibit the dopamine transporter, leading researchers to consider the dopamine transporter a primary candidate gene for ADHD (Cook, Stein, Krasowski, Cox, Olkon, Kieffer, & Leventhal, 1995). A study examined the relationship of the dopamine transporter Gene (DAT 1) to symptoms of internalizing disorders. Please note the following pertinent definition: Allele is

one of two or more alternative forms of a gene occurring in corresponding sites on homologous chromosomes, any two of which may be carried by a given individual and which determine alternative characters in inheritance (Dorlands Medical Dictionary, 1974). The research performed has indicated the following:

1. Levels of both hyperactive-impulsive and inattentive symptoms increased with the number of DAT1 10 repeat alleles alternative characters.
2. In analyses of genetically discordant siblings, the siblings with the greater number of DAT1 repeat alleles had heightened symptoms than their co-siblings with fewer than 10-repeat alleles.
3. Using the transmission disequlibrium test, the 10-repeat allele was preferentially transmitted from heterozygous parents to children who exhibited ADD/ADHD behavior patterns. (Rowe, et. al, 1997)

These studies have genetic implications for the transmittal of ADD/ADHD characteristics. These studies suggest that genes encode or serve as the blueprint for dopamine receptors and transporters and are active in the prefrontal cortex and basal ganglia (Barkley, 1998; Gibbs , 1998). The controversy of nature vs nurture will continue to be examined as researchers seek the etiology of ADD/ADHD.

Assessment and Treatment of ADD/ADHD

Current Assessment Procedures

Currently, in order to insure that evaluation provides a reliable, accurate, and valid diagnosis, appropriate assessment instruments should be used (Barkley, DuPaul, & McMurray,

1990; Shaywitz, S.E., Holhn, Marchione, Sadler, & Shaywitz, 1992; Witt, Heffer & Pfeiffer, 1990). Assessment procedures currently in place are behaviorally based and usually consist of some form of rating scale such as the Conners Behavior Rating Scale, the ADD-H Comprehensive Teacher Rating Scale (ACTeRs, the Attention Deficit Disorder Evaluation Scale, the School and Home Situations Questionnaire and the Achenback Child Behavior Checklist, and the latest, the Gordon Diagnostic System, an instrument reported to use tasks that are sensitive in the identification of impulsive, inattentive qualities of hyperactive children(Goldstein & Goldstein, 1992; Ingersoll & Goldstein, 1993). These measures are scored by a trained professional, based on a standardized rating system, to determine if a child is at risk for an ADD/ADHD diagnosis and needs referral to a clinical psychologist and/or the primary care physician.

Other diagnostic tools include: a complete developmental history, medical history, detailed observation performed and reported by the child's teacher, school psychologist and/or a behavioral consultant. This will help rule out other factors such as "Goodness of Fit" problems which the child is mismatched with his environment or home expectations (Watson, 1998).

Traditional Treatment

From a neurological perspective, medication is the traditional treatment for ADD/ADHD, with Ritalin being the most prescribed medication. The prescription of Ritalin has increased more than sevenfold in the last 8 years and 90% of it is consumed in the U.S. Ritalin is prescribed based on the medical hypothesis that ADD/ADHD stems from an inadequate supply of dopamine and norepinephrine in the brain. and Ritalin boosts the dopamine levels. (Gibbs, 1998). In November of 1998, the National Institute of Mental Health (NIMH), met to determine a consensus concerning the use of Ritalin. They concluded that:

1. Ritalin clearly works in the short term for reduction of ADHD symptoms
2. More studies are needed on the long term effects of Ritalin on academic or social performance.
3. Ritalin may stifle appetite and delay growth in some children.
4. A positive response to Ritalin does not automatically indicate ADHD. Stimulants can sharpen anyone's focus.
5. Ritalin is not a panacea. It won't boost IQ or take away learning disabilities that affect 15% of children with ADHD.
6. Preliminary evidence suggests that the brains of children with ADHD differ from their peers. Researchers are not sure if this is due to normal variation or is truly biochemical.

(NIMH, 1998)

Barkley (1998), suggests that psycho-stimulant medical compounds such as Ritalin improved the behavior of 70 to 90 percent of children, ages 5 and older, who are diagnosed with ADHD. Barkley suggested that medications not only reduced impulsitivity, restlessness and distractibility but also helped retention of academic information. Drug therapy variables include the doctors' expertise, child's age, severity of the behavior, dosage supervision, genetic history, anxiety levels, and the child's emotional stability (Ballard, 1997).

Other Treatment Approaches

Watson (1998), an Educational Psychology Professor at Mississippi State University, advocates a behavioral intervention approach for children exhibiting typical ADD/ADHD behaviors. This approach includes observation of the child in all settings, charting of antecedents

to the behavior, development of a plan of reinforcement of desired behaviors, modeling desired reinforcement procedures for the parent/teacher, and careful monitoring of the implementation. Parents and teachers can aid children by anticipating events, breaking tasks down into smaller and immediate steps, and modifying location and stimulus within the classroom. The purpose is to externalize time, rules and consequences in order to replace internal forms of information, rules and motivation (Jensen, 1998). These approaches modify the environment to increase the desired behaviors.

Future research into the possible genetic and neurological etiologies of ADD/ADHD should lead to additional treatments and a more specific means of identifying the disorder of ADD/ADHD. When dealing with the brain, scientists and physicians need to develop treatment strategies that produce long term positive effects. Possible future treatments include the development of new medications and better implementation of current medication, a reduction in the diagnosis of ADD/ADHD percentage, due to new medical tools such as brain scans, and recommendations for implementation of behavioral assessment procedures which lead to appropriate interventions.

Conclusions

A review of the history of the diagnosis and treatment of ADD/ADHD and the possible neurological and genetic etiologies of this disorder suggests the following conclusions.

1. ADD/ADHD is a true behavior disorder that is probably present in 5-9 percent of the school-age population
2. Ritalin, while sometimes effective, is over-prescribed and lacks research on side effects.
3. Neurological studies involving brain scans indicate certain peculiarities of the brain among approximately 70% of the children diagnosed with ADD/ADHD.

4. Family and twin studies suggest a genetic etiology of ADHD, possibly helping predict the disorder and leading to early treatment.
5. The best assessment approach leading to treatment is one that includes a complete developmental history, medical history, classroom/home observations, traditional standardized tools, and referral to medical and behavioral specialists.

More research is needed in the areas of: neurology and genetics as etiologies of ADD/ADHD, the long-term effects of Ritalin upon children and adults, and behavioral techniques to modify ADD/ADHD behaviors. Further research offers hope for more effective treatments, improvement of the quality of relationships with others, and discovery of the extent of cormobidity with other disorders.

APPENDIX

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APPENDIX

ADHD Checklist

I. DSM-IV Diagnostic Criteria for ADHD

A. 6 or more of the following symptoms of inattention or hyperactivity-impulsivity, which have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

___fails to give close attention to details

___makes careless mistakes in schoolwork or other activities

___has difficulty sustaining attention in tasks or play activities

___does not seem to listen when spoken to directly

___does not follow through on instructions, fails to finish schoolwork, chores, or duties in the workplace

___has difficulty organizing tasks or activities

___avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)

___loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)

___easily distracted by extraneous stimuli

___forgetful in daily activities

Hyperactivity

___fidget with hands or feet, squirms in seat

___leaves seat in classroom or in other situations in which remaining seated is expected

___runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)

___has difficulty playing or engaging in leisure activities quietly

___is often "on the go" or often acts as if "driven by a motor"

___talks excessively

Impulsivity

____blurts out answers before questions have been completed

____has difficulty awaiting turn

____often interrupts or intrudes on others (butts into conversations or games

B. Some symptoms must be present before the age 7 years
C. Some impairment from symptoms is present in 2 or more settings
D. Clear evidence of clinically significant impairment in social, academic, or occupational functioning
E. Symptoms do not occur exclusively during the course of other mental disorders

DSM-IV (1994)
***Operative word OFTEN